Issued May 18, 1907.

U. S. DEPARTMENT OF AGRICULTURE,

BUREAU OF ANIMAL INDUSTRY.—CIRCULAR NO. 109.

A. D. MELVIN, CHIEF OF BUREAU.

QUAIL DISEASE IN THE UNITED STATES.

(A Preliminary Report.)

By GEO. BYRON MORSE, M. D., V. S.,
Assistant in Bacteriology and Pathology, Division of Pathology.

A highly contagious and rapidly fatal disease has recently prevailed among quail in this country. New to the United States, or at least hitherto unrecognized, its enzootic character, already manifest in some localities, makes it a matter of grave concern to naturalists, ornithologists, members of Audubon societies, sportsmen, and owners of hunting preserves.

English sportsmen know too well the gloom cast over grouse hunting by the dreaded grouse disease. It is hardly possible with the data at hand to claim identity of the British disease of grouse with the fatal malady now attacking the quail of this country. However, the near relationship of the birds—all being members of the same family, Tetraonidæ; the close resemblance in several essential features of the two diseases; the fact that Klein's bacillus of grouse disease (*Bacillus scoticus* Migula) and the bacillus incriminated in this report are, if not identical, closely related members of the same group of bacteria (*Bacillus coli*), all combine to make a brief review of the history of grouse disease exceedingly appropriate.

HISTORICAL NOTE ON GROUSE DISEASE.

The moorlands of England and Scotland have paid heavy tribute to disease. Doctor MacDonald, writing in 1883, said: "It is now eighty years since the alarm of grouse disease was sounded in this country." As far back as 1817 the Sporting Magazine, in its issue for October of that year (as quoted by the London Field [a]), said: "An extraordinary disease has lately spread more havoc among the grouse in the north of Scotland than the double-barreled guns

[a] The Field, London, June 16, 1877, Vol. XLIX, No. 1277, p. 705.

30312—No. 109—07 M

of the numerous sportsmen. The birds are found dead on the hills in great numbers."

The disease was made a subject of investigation as early as 1828. The Greenock Advertiser of that year contained the following: "Mr. Wallace, of Kelly, had numbers of diseased grouse dissected and found them generally infested with tapeworms." The London Field (cited above), which reprinted this extract, added that "Mr. Wallace attributed this destructive malady and the occasional scarcity or plentifulness of game generally to one and the same cause, viz, a continuance of damp and wet weather, with little sunshine, during the spring and summer months."

In the course of the discussion aroused over the disease, various causes were ascribed for its origin. It was claimed that the grouse were suffering from lead poisoning, having eaten the shot which lay in enormous quantity over the moors from the previous season; that they had drunk poisonous sheep dip; that they had been enfeebled from gunshot wounds; that they had suffered from overcrowding on a breezy hillside; that they had degenerated from want of crossing; that the blight of the heather by the east wind had deprived them of proper nourishment, and finally that (according to Doctor Farquharson, the chief exponent of this theory) it is a contagious epidemic like scarlet fever, diphtheria, or typhoid fever.

The name of T. Spencer Cobbold is closely associated with the history of grouse disease by reason of his promulgation of the parasitic theory. This well-known writer on parasites believes that he found the cause of the affection in *Strongylus pergracilis* Cobbold, a minute nematode which he found in great numbers in grouse dying of grouse disease.[a]

The finding of tapeworms and strongyles in large numbers in otherwise healthy grouse that showed no signs whatever of grouse disease left the question, in the minds of many, still open. The London Field in its issue of May 21, 1887 (Vol. LXIX, p. 683), announced its offer to have careful investigation of diseased birds made by Dr. E. E. Klein, principal of the Brown Institution. As the result of this investigation Klein made three reports to The Field embodying the findings of his researches. For the purposes of this review they may be briefly summarized as follows:

Post-mortem findings showed the birds to be as a rule very emaciated. The characteristic lesions were congestion of the lungs, liver, and kidneys, with small superficial necroses of the liver, and patchy redness of the intestines. (The presence of pneumonia has been noted frequently enough to call forth the name since then of pneumo-

[a] T. Spencer Cobbold, The Grouse Disease, "The Field" Office, London, 1873, p. 27.

enteritis of grouse.) The heart was filled with coagulated blood. Bacteriologic investigation resulted in the isolation, cultivation, and successful inoculation of a rather polymorphic micro-organism which, according to Klein's description, and the further results obtained by Theobald Smith and Lignières, is proven to be a variant of *Bacillus coli.*

The public and the scientific world have very generally accepted the findings of Klein, and hence we may say that grouse disease is an infectious disease of grouse (*Lagopus scoticus*) caused by a microbe of the *B. coli* group, and characterized by congestion of the lungs, necrotic areas in the liver, and patchy redness of the intestines.

DEFINITION OF QUAIL DISEASE.

Assuming, until we have further data upon grouse disease, the nonidentity of the two diseases, the plague appearing among bobwhites in this country may be thus defined:

Quail disease (*Colibacillosis tetraonidarum*) is an infectious disease of the grouse family, caused by a microbe of the *B. coli* group, and characterized by congestion of the lungs, focal necroses of the liver, and intestinal ulceration.

HISTORY OF THE EPIZOOTIC IN THE UNITED STATES.

On April 3, 1906, three dead bobwhites were received from a dealer in Washington. Accompanying the birds was the statement that these were among the last of a large number that had been steadily dying off. At night the whole flock would appear well; in the morning several would be dead. Upon necropsy these birds showed the alterations described later under "Post-mortem appearances." Bacteriologic investigation resulted in the isolation and cultivation of a virulent *Bacillus coli*, whose high percentage of gas formation in dextrose and lactose bouillons gave it a marked resemblance to Klein's bacillus of grouse disease as studied by Theobald Smith.

Some time later this dealer received from Wichita, Kans., two dozen blue quail. Nearly all the birds were dead on arrival. Although no post-mortem work was attempted, the dealer, a most competent man, of thorough scientific training, avers that he was able to recognize the disease by the characteristic odor of the droppings. He further states that the previous year he had received shipments from Birmingham, Ala., one of which had a mortality of 100 per cent.

In May, 1906, this office received a letter from Boston, Mass., regarding disease among quail there. This letter contained an inclosure from an eminent Boston pathologist noting the same findings that are described below. On account of decomposition no bacterio-

logic work was attempted. It was stated, however, that no ameba could be found.

On January 2, 1907, a letter was received from Worcester, Mass., in which the writer deplored the fact that disease was the great hindrance to the rearing of ruffed grouse in that section. Further investigation revealed the fact that bobwhites were also included in this complaint. The birds with which breeding work had been undertaken were obtained from Alabama.

On February 11, 1907, the Washington dealer sent to this laboratory two dead bobwhites with the statement that of 65 just received from Alexander City, Ala., 34 were dead. Post-mortem examination was made of not only the 34, but also of nearly all the others that died. The same lesions were revealed.

On February 23, 1907, there were received from the same person 15 birds—8 Kansas bobwhites and 7 scaled quail ("cotton-top" or blue quail of Texas). These quail had been for several months on the place and had appeared perfectly well. The infected shipment of February 11 was brought to the entrance of the building where the 15 were kept. In ten days the disease broke out among them and in two days all were dead.

On February 25, 1907, two crates of live bobwhites were received from this gentleman. This represented a shipment of 5 dozen birds from Alexander City, Ala., to take the place of the other 5 dozen that had died. Upon arrival in the city 2 were dead. These were examined by Dr. John R. Mohler, Chief of the Division of Pathology, and pronounced by him to be cases of quail disease. The shipment was immediately transferred to this laboratory. Within eight days all had died except 3, which 3, removed to fresh cages, continue at the present writing apparently perfectly free from disease.

On February 26, 1907, another dealer in Washington received a shipment of 53 bobwhites from Indian Territory, but which had come by way of Wichita, Kans. There exists some uncertainty as to whether or not they were recrated at Wichita, but, as will be recognized later, this question is of no moment, for the disease is in Indian Territory. On arrival 12 of this lot were dead and 4 died subsequently. These birds showed the lesions of quail disease. The rest of the birds were sent to Mr. H. H. Dodge, custodian at Mount Vernon, Va. Mr. Dodge has since sent to this laboratory 4 of these birds, dead from the same disease.

Another shipment of bobwhites from Marlow, Ind. T., consigned to the first-mentioned dealer arrived March 4, 1907. Of this lot 26 were dead, and post-mortem examination of 20 of them proved the existence of the disease and the presence of the causative bacillus.

March 5, 1907, there was received for examination one female

California quail, forwarded from Elizabeth, Pa. Putrefactive changes had advanced too far to permit bacteriologic examination, but the lesions pointed strongly to quail disease. However, it is of interest to note that the lesion-complex was that which is peculiar to grouse disease rather than the disease among quail in this country, namely, pneumonia, superficial necroses of the liver, and congestion of the intestines.

March 17, 1907, the second-mentioned Washington dealer received from Wichita, Kans., a lot of 48 bobwhites. Upon arrival 6 were found dead of quail disease. The balance were shipped to Mount Vernon, whence 2 have been returned to this laboratory for autopsy.

March 21, 1907, a sharp-tailed grouse was received for autopsy from the first-mentioned Washington dealer. A most perfect picture of quail disease was presented by this bird. The next day 2 more sharp-tailed grouse died with the same lesions, and another is reported sick.

Word has also been received of the disease occurring at Yarmouth, Nova Scotia. These bobwhites came from Alabama.

GEOGRAPHICAL DISTRIBUTION.

The facts recorded in the previous section constitute all that we know at present concerning the geographical distribution of quail disease. It is certain, however, that the disease is occurring wherever shipments of bobwhites or other quail are being received from those distributing centers which have become infected. For instance, if we hear of a person receiving quail from one of the infected points in Alabama, we shortly afterwards hear of his lot of birds dying with disease which proves to be quail disease.

According to the above-recorded facts, quail disease exists or has occurred in Alabama, Kansas, Indian Territory, Washington, D. C., Mount Vernon, Va., Elizabeth, Pa., Boston and Worcester, Mass., and Yarmouth, Nova Scotia.

KNOWN CENTERS OF INFECTION.

By this is meant collecting and distributing points which have been proved infected by the fact of birds shipped from those points having died en route or immediately after arrival and the presence of the disease demonstrated by post-mortem examination. Such centers of infection are Alexander City and Dadeville, Tallapoosa County, and Birmingham, Jefferson County, Ala.; Wichita Kans., and Marlow, Chickasaw Nation, Ind. T.

SPECIES AFFECTED.

Post-mortem examination has up to the present time demonstrated the presence of quail disease in the common bobwhite (*Colinus vir-*

ginianus), the California quail (*Lophortyx californicus vallicola*), the Gambel quail (*Lophortyx gambeli*), the mountain quail (*Oreortyx pictus*), the scaled quail, called also "cotton-top" or blue quail (*Callipepla squamata*), and the sharp-tailed grouse (*Pediocætes phasianellus campestris*).

Had the disease remained limited to the quail only it would have been serious enough, but the recent demonstration of its transmissibility to sharp-tailed grouse is nothing less than appalling in its suggestion of widespread fatality among game birds. This fact calls for the most energetic action on the part of all lovers of sport and all who appreciate the economic importance of these birds, in noting and reporting every outbreak of the disease, and, if possible, tracing its origin.

SYMPTOMS.

The period of incubation appears to be about ten days. The disease first manifests itself by dullness and a tendency to sit in the corner of the cage with feathers fluffed. The food is neglected, and generally in two or three days (before diarrhea has developed or emaciation made its appearance) the bird is dead. Sometimes the disease runs a more chronic course, and, though diarrhea is rarely very marked, emaciation becomes extreme.

POST-MORTEM APPEARANCES.

On picking the birds one is surprised to find them presenting, as a rule, such a fine appearance. Their plump, meaty breasts of perfectly normal hue contrast strongly with what is seen upon opening the body cavity. There are exceptional cases, however, exhibiting great emaciation. Upon exposing the viscera, the note that is made usually reads: "Lungs slightly congested; liver congested and presenting a few small areas of superficial necrosis; intestines studded with minute ulcers." These may be called the salient features of the disease, the characteristic lesions that, immediately on opening the carcass, attract the attention of the observer, be he layman or scientist.

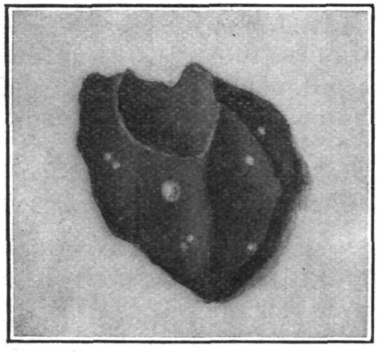

FIG. 1.—Liver of bobwhite showing a quite frequent form of focal necrosis.

Sometimes, though rarely, the lungs exhibit areas of consolidation, represented by small, dark-red spots. Often the liver presents no necrotic areas, is merely congested; on the other hand, sometimes the

destruction of tissue will involve a large portion of a lobe, in depth as well as in extent of surface. The spleen is always congested, sometimes enlarged, sometimes quite dark. The constant feature, however, is the intestinal lesion. This may be recognized, generally, by the presence here and there, throughout the length of the intestine, of minute to large pinhead-sized areas of necrosis which are seen through the wall of the intestine as small, yellowish spots. Sometimes there is merely what Klein has described in his report on grouse disease as "patchy redness."

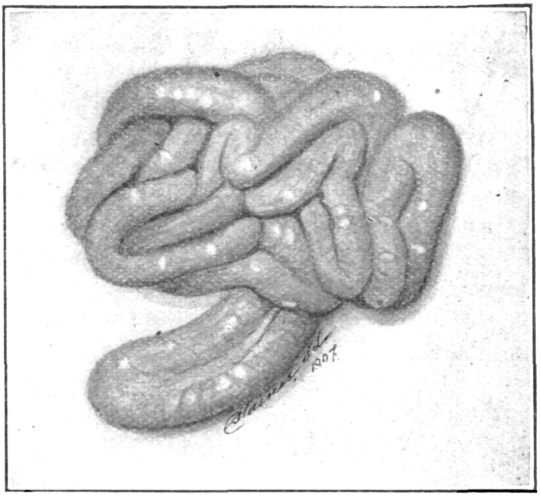

FIG. 2.—Intestines of bobwhite showing characteristic form of lesion.

Again, there may be distinct, minute erosions; also, as first mentioned, there may be focal necroses covered with a croupous exudate. Quite often the necrotic process has gone on until a true ulcer is formed, sometimes even penetrating the wall of the gut. In the event of this last condition obtaining we may expect to find little yellowish masses of necrotic material coating the intestines or the walls of the abdominal cavity.

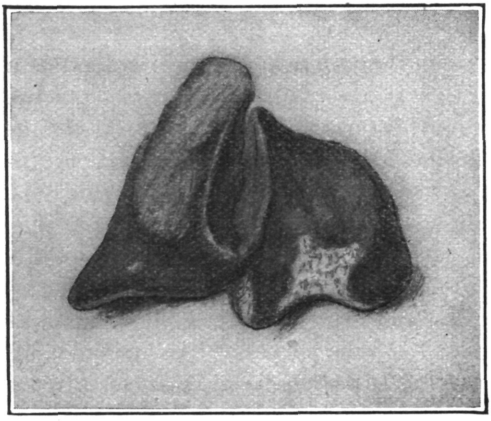

FIG. 3.—Liver of bobwhite showing extensive necrosis.

INVESTIGATION OF THE CAUSE.

As already stated, in the investigation of the outbreak of April, 1906, an indication was obtained as to the nature of the cause of the present epizootic. This pointed directly to Klein's bacillus of grouse disease, *B. scoticus*, Migula. However, bearing in mind the differences in description of the two diseases, search was made for causes other than that of so-called grouse disease. A superficial resemblance, in some cases at

least, to the infectious entero-hepatitis or blackhead of turkeys suggested looking for amebæ; but no ameba could be found. In view of this likeness it might be well to note certain points of difference. The liver is affected in a much less proportion of cases in quail disease than in infectious entero-hepatitis; the contour of the liver lesions in the latter disease is much more regular than in quail disease; enlargement of the liver is not the characteristic in quail disease that it is in blackhead. In blackhead it is the ceca and never the intestines that are affected; in quail disease the intestines proper, from gizzard to vent, are studded with ulcers, the ceca being diseased in only about 40 per cent of the cases. Finally, as stated, there is no ameba present. There are bodies present in the lesions which possess apparently the same size and structure as the *Ameba meleagridis*, described by Smith, but the writer has no hesitation in saying that they are degenerated and degenerating nuclei of tissue cells.

On the other hand, bacterial research gave different and far more definite results. With striking unanimity all diseased birds yielded a bacillus of the *B. coli* group. Sowings made from blood, lungs, liver, and intestines gave rise to a rather actively motile rod-shaped organism, appearing often as paired ovals, single or paired rods with rounded ends, and even filaments. Polar staining was quite constant, staining by Gram negative, and no spores were ever detected. Growth was independent of the presence or absence of oxygen. Its growth on gelatin, the character of gas formation in glucose, lactose, and saccharose bouillons and its milk coagulating properties prove it to be a form of *Bacillus coli*, an intestinal micro-organism of widely extended pathogenic powers.

The bacillus can not always be cultivated from the heart blood of a bird dead of quail disease, nor can it always be cultivated from the lungs. It can usually be isolated from the liver in which lesions exist, and always from the affected intestines. Thus far in the investigation the organism has not proved pathogenic for chickens, pigeons, or rabbits. It has produced death in mice and guinea pigs, with the characteristic lesions. As was the case with Klein in his grouse disease investigations, great difficulty has occurred in securing birds known to be free from exposure. With the small amount of material available we have still been successful in inoculating birds with cultures, although feeding experiments have as yet proved negative.

TREATMENT.

At present no curative treatment can be described with promise of success. As brought out under symptomatology, the subtle invasion of the disease and its seemingly sudden development with quickly

ensuing death preclude the possibility of success in individual treatment. Nevertheless there are cases that appear at post-mortem to have been chronic, and hence in individual cases it might be well to institute treatment by intestinal antisepsis; for instance, small doses of calomel (one-tenth of a grain). For reasons cited in the section below, immediate change of location, with disinfection of cages, water pans, feed trays, etc., should be made.

PREVENTION.

It is difficult at this stage of the investigation to speak dogmatically on this subject. Certain facts combine to establish at least one definite line of procedure. The disease has been recognized only in birds in the state of captivity. It can not, however, be positively asserted that it does not occur among those in the wild state. Wild birds living in their natural state under ordinary conditions are not called upon to develop resistance to the bacteria that swarm in crowded habitats. Let the ordinary conditions of food supply be disturbed by circumstances that curtail the amount of ground available to the birds, and at once attention is called to the mortality among them, and investigation shows bacterial disease.

When birds are caught and maintained in captivity, either in the small inclosures of the dealers or even in the less small grounds of developing preserves, the same conditions as above mentioned obtain and great mortality from bacterial disease results. Therefore the following precautions are urged upon all who have to do with not only quail but all wild birds kept under artificial conditions.

In the case of caged birds, the cages should be kept scrupulously clean and the food kept in containers that will prevent the contamination of the food supply by the feces. Where birds are kept in good-sized inclosures frequent change of location is necessary, the used ground being treated with lime, plowed deep, and allowed to sweeten.

Collectors and shippers should regard the disease as a menace to their business. Crates of such simplicity and cheapness of construction should be employed as would permit their being burned after using once. The practice of shipping back the "empties" is most baneful. When a shipper has once had the disease on his place all fresh birds should be received in another building at as great a distance as possible from the inclosure where the sick birds were kept. The infected ground or building should then be disinfected as above directed.

The principles mentioned above, while of general application, are specially appropriate to the prevention of quail disease. It is a

disease due to *Bacillus coli*. This is an intestinal germ, whose presence in food or water supply bespeaks contamination with feces. Hence, by cleanliness, by disinfection, by change of ground such contamination should be prevented.

CONCLUSION.

A fatal disease at present menaces the quail interests of this country. In time of outbreak, mode of attack, general character of lesions, and causative agent it is singularly like the grouse disease of England. Actually unknown in this country until the past year, it demands the active interest of all lovers of bird life in order that it may be better understood with a view to its control and prevention.

Some questions pertaining to the problem that require an answer are as follows:

1. Is the disease due to a specific contagium introduced into this country from without, as, for instance, grouse disease of England, or is it a disease of overcrowding that may arise in any section of the country whenever the birds are brought under artificial conditions of life?

2. Are there, as in grouse disease, two outbreaks—one, the acute, in spring, and the other, a subacute, or chronic form, marked by emaciation, in the fall?

3. Does the disease seem to occur spontaneously in sections of the country other than those named under "Known centers of infection?"

4. How widespread is the disease? Is its geographical distribution, as outlined in a previous section, limited to those localities which have received shipments from the known centers of infection?

5. Does the receipt of a shipment of birds result in the infection of birds heretofore healthy? The facts recorded in this circular point to this, but further evidence will be of value.

6. If the preceding question is answered affirmatively, what period of time elapses between the receipt of such infected birds and the outbreak among the old stock? In other words, what is the period of incubation?

7. Besides the cases mentioned in this paper, are there other persons who have received shipments of bobwhites or other quail from Alabama, Kansas, or Indian Territory, only to have them shortly die from disease?

These and similar questions press for answer before it can be claimed that we know the disease and are therefore prepared to combat it intelligently. Any information that will throw further light upon the problem will be welcomed by the Bureau of Animal Industry. Upon those who are interested in this chief of American

game birds it can not be urged too strongly that they give immediate notification of the existence of disease among the quail of their sections of the country. Wherever possible a few of the dead birds should be wrapped in cloths saturated in 5 per cent carbolic acid solution or 10 per cent formalin and forwarded immediately to the Bureau of Animal Industry, Division of Pathology, Washington, D. C., for verification of the disease. The men who are acquainted with the birds and their haunts should note carefully everything connected with the outbreaks of quail disease. The layman and the scientist must work together if any successful defense is made against this destructive malady.

Approved:

JAMES WILSON,
Secretary of Agriculture.

WASHINGTON, D. C., *April 8, 1906.*

O